

# ARTERIAL HYPOXEMIA IN ACUTE TROPICAL PULMONARY EOSNOPHILLIA

VIJAYAN, VK., KUPPURAO, KV., VENKATESAN, P., AND PRABHAKAR, R.

Cardio-Pulmonary Medicine Unit, Tuberculosis Research Centre, Chetput, Madras-600 031

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## ABSTRACT

Arterial oxygen tensions were estimated in 48 acute tropical eosinophilic patients. Twenty (42%) had  $\text{PaO}_2$  of less than 80 mmHg, though 18 had only mild hypoxemia ( $\text{PaO}_2$  -70-80 mmHg). A smoker had  $\text{PaO}_2$  of less than 60 mmHg. The single breath carbon monoxide transfer factor (TLCO) was lowered in 42 (88%) patients. The  $\text{PaO}_2$  correlation with both  $\text{FEV}_1\%$  and TLCO percent predicted was not strong. Obstructive ventilatory and diffusion defects may not be the main mechanisms of hypoxemia in these patients.

## Introduction

Tropical Pulmonary Eosinophilia (TPE) is a syndrome characterised by cough, dyspnoea, wheezing, diffuse reticulonodular opacities in chest skiagram and marked peripheral blood eosinophilia(1-3). The syndrome is thought to be due to immunologic hyper-responsiveness to filarial human parasites, *Wuchereria bancrofti* and *Brugia malayi*(4). Previous pulmonary function studies had shown that there is a combination of obstructive and restrictive ventilatory defects in TPE (5-9). However, arterial blood gas studies are scarce and published studies had shown a mild arterial oxygen desaturation(10-13). Udwadia(2) suggested that hypoxemia in longstanding cases of TPE was due to impairment of diffusion. Poh(11) felt that hypoxemia in Acute TPE might be due to airway obstruction and Ray(13) hypothesised that it might be due to ventilation-perfusion inequalities in the lung. These studies reveal that the exact mechanism of arterial hypoxemia in TPE is not well understood. The purpose of the present study is, therefore, to explore the mechanisms of arterial hypoxemia in acute tropical eosinophilia.

## Material and Methods

Fortyeight consecutive patients with recent onset of symptoms of one week to six months duration and fulfilling the inclusion criteria of residence in the endemic area of Madras city, respiratory symptoms Such as cough, dyspnoea and wheezing, chest skiagram opacities, lung function abnormalities, peripheral blood eosinophilia of  $>2000$  cells/cu.mm., high serum titres of anti-filarial IgG and a response to diethyl carbamazine therapy were included in the study(4). All the patients in this study were cases of

acute TPE of recent onset and none had acute exacerbations of chronic TPE. Evaluation of each individual included detailed history, physical examination, chest skiagram, blood and stool examinations for the presence of other parasites and IgG filarial antibody determinations.

Pulmonary function tests such as Forced Expiratory Volume in 1 second ( $\text{FEV}_1$ ), Forced Vital Capacity (FVC),  $\text{FEV}_1/\text{FVC}\%$ , and Single Breath Carbon Monoxide Transfer Factor (TLCO) were carried out using P.K. Morgan (U.K.) Transfer Test Model C. A minimum of three consistent readings in the case of spirometry and two in the case of TLCO measurements were obtained, and the highest value obtained was used for analysis. All the values were expressed in B.T.P.S. If the ratio of  $\text{FEV}_1/\text{FVC}$  is less than 75% (14) the patient is classified as having obstructive ventilatory defect.

Pulmonary function studies were carried out in 250 normal subjects in our laboratory, using P.K. Morgan (U.K.) Transfer Test Model C in order to establish regression equations for predicting normal pulmonary function values in Southern Indian subjects. The predicted values based on age, height and weight for this study were obtained from these regression equations (in preparation). The measured values of TLCO are considered normal, if it is within  $\pm 15\%$  of the predicted (15).

The arterial blood gases were determined using Radiometer (Copenhagen) ABL III apparatus. The direct arterial puncture, using special pyrogen free Ethylene Oxide sterilised Arterial Blood Sampler (B109) provided by Radiometer was done either from the brachial artery or the radial artery with the

patient in the supine position and respiring room air. The blood gases were determined immediately after the arterial puncture. In our laboratory, we had carried out arterial oxygen tension estimation in 97 normal individuals and the mean arterial oxygen tension in those below 40 years of age was  $97.3 \pm 8.4$  mmHg (unpublished observations). Since Madras is at sea level (Barometric pressure-approx. 760mmHg), and all except two of the patients in this study were aged less than 40 years, those who had arterial oxygen tension  $\leq 80$  mmHg were classified as having hypoxemia.

### Results

There were 43 males and five females in the study. All the females were non-smokers. Among the males, 20 were non-smokers and 23 were smokers. The physical characteristics of the patients are given in Table 1.

**TABLE-1**

#### PHYSICAL CHARACTERISTICS

Factor	Mean $\pm$ SD
Age (years)	$24.0 \pm 7.5$
Height (cm)	$160.9 \pm 7.7$
Weight (kg)	$44.9 \pm 8.3$

The mean age was  $24.0 \pm 7.5$  years (Range 12-48 years) and the mean height was  $160.9 \pm 7.7$  cm. As it was observed that there were no significant differences in mean values of FEV<sub>1</sub>/FVC%, TLCO% predicted and PaO<sub>2</sub> between smokers and non-smokers, the data were amalgamated for further analysis of results.

The pattern of pulmonary function abnormalities are given in Table 2. 14 patients (29%) had a combination of obstructive ventilatory (FEV<sub>1</sub>% less than 75%) and diffusion defects, (TLCO <85 percent of predicted) 28 (58%) had diffusion defect alone and 3 (6%) had obstructive ventilatory defect alone. Thus, a total of 42 patients (88%) had diffusion defect, alone or in combination with obstruction. Obstructive ventilatory defect, alone or in combination with diffusion defect was seen in 17 (35%) patients.

**TABLE-2**

#### PATTERN OF PULMONARY FUNCTION

Obstruction	Diffusion defect	Obstruction and Diffusion defect	Normal
3 (6%)	28 (58%)	14 (29%)	3 (6%)

82% of the patients with obstruction (14 out of 17) had mild obstructive ventilatory defect (FEV<sub>1</sub>/FVC% ranging from 60-75%). However, 52% of patients with diffusion defect (22 out of 42) had moderately severe diffusion defect (TLCO% predicted ranging between 41-70%).

Arterial hypoxemia was observed in 20 patients (42%). Table 3 shows the severity of hypoxemia observed in these patients.

**TABLE-3**

#### SEVERITY OF ARTERIAL HYPOXEMIA

Severe ( $<60$ mmHg)	Moderate (60-70mmHg)	Mild (70-80mmHg)	Normal! ( $>80$ mmHg)
1 (2%)	1 (2%)	18 (38%)	28 (58%)

Most of the patients (90%) with reduced arterial oxygen tension (18 out of 20) had only mild arterial hypoxemia while one patient was in hypoxic respiratory failure range (PaO<sub>2</sub> 60 mmHg and PaCO<sub>2</sub> 38.8 mmHg). He was a smoker.

To identify the factor responsible for arterial hypoxemia in Acute TPE, arterial oxygen tension was correlated with FEV<sub>1</sub>/FVC% and TLCO% predicted (Fig. 1 and 2). The positive correlation of PaO<sub>2</sub> with FEV<sub>1</sub>/FVC% ( $r = +0.361$ ,  $P < 0.01$ ) and with TLCO% predicted ( $r = +0.353$ ,  $P < 0.05$ ) was not strong though significant statistically.

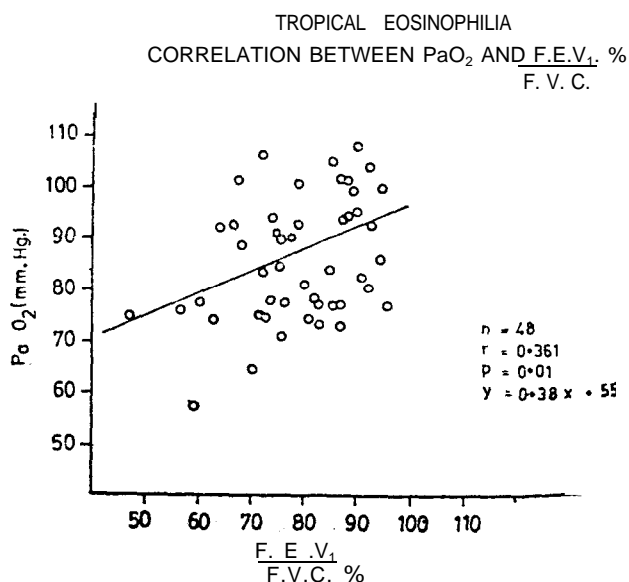
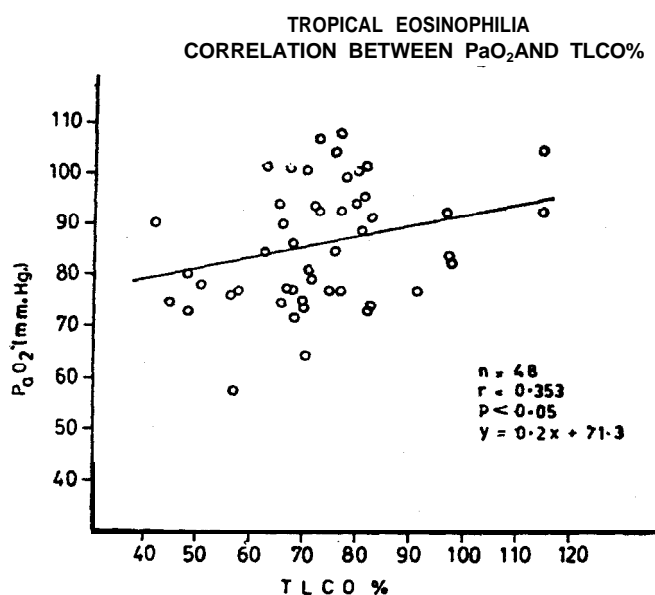


Fig. 1



### Discussion

Previous studies (5-9) had shown that there was combined obstruction and restriction in Tropical Pulmonary Eosinophilia. Mild arterial oxygen desaturation in Tropical Eosinophilia was reported by Udwadia(2), Mathur(10), Poh(11) and Ray (12,13). However, none of these investigators had reported the actual arterial oxygen tension determinations in acute TPE and our observation of mild hypoxemia corroborates the mild arterial oxygen desaturation observed by these investigators. The observation of hypoxic respiratory failure in one of our patients points to the fact that severe hypoxemia can occur in a small proportion of patients with acute TPE (perhaps aided by the smoking habit).

Poh(11) was the first person to report a decrease in Transfer Factor by steady state end-tidal sampling method. We had also observed that, in 88% of patients with acute Tropical Pulmonary Eosinophilia, there was a reduction in Transfer Factor for carbon monoxide by single breath method. Poh(11) had also observed in a study of 15 patients that six patients with obstructive and restrictive pattern were hypoxemic and suggested that hypoxemia is likely to be due to airway obstruction. Ray(13) had put forward the hypothesis that ventilation-perfusion mismatch may be responsible for arterial hypoxemia. The weak correlation of PaO<sub>2</sub> with FEV<sub>1</sub>/FVC% ( $r = +0.361$ ) and with TLCO% predicted ( $r = +0.353$ ) shows that obstructive ventilatory and diffusion defects may not be the main mechanisms of hypoxemia in acute Tropical eosinophilia. Further studies are required to know

whether ventilation perfusion mismatching is the mechanism of hypoxemia in acute Tropical Eosinophilia.

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